Complications of Hepatic Chemoembolization

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ABSTRACT

Transarterial hepatic chemoembolization continues to evolve as an integral therapy for unresectable primary and secondary liver tumors. Despite relatively low morbidity, major complications may be seen. This article provides an overview of the spectrum of vascular and nonvascular complications related to this therapy.

KEYWORDS: Hepatic chemoembolization, hepatocellular carcinoma, liver metastases, complications

Objectives: On completion of this article, the reader should be familiar with the spectrum of vascular and nonvascular complications associated with hepatic chemoembolization.
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Since its introduction over 25 years ago as a treatment for unresectable hepatocellular carcinoma, transarterial hepatic chemoembolization (TACE) has undergone worldwide dissemination to become an established therapy for both primary and secondary hepatic malignancies. Although variations exist in the technique of TACE, these technical differences share a common set of complications. Major complications occur in ~5% of patients, with a risk of death of ~1%. The interventional radiologist needs a thorough understanding of the complications unique to TACE and the time frame in which these complications may develop. This article reviews the vascular and nonvascular complications associated with TACE.

VASCULAR COMPLICATIONS

Vascular complications of TACE relate to arterial access (puncture site complications, placement of a catheter in the hepatic artery, injury to the hepatic artery itself) and nontarget embolization are reviewed.

Access Site Injuries

Because TACE can be performed through small-caliber diagnostic (4 to 5 French) catheters and microcatheters, placement of large-diameter sheaths at the access site is seldom necessary. Therefore, complications related to puncture site access are uncommon. Access site hematoma is most frequently encountered and occurs in ~2% of patients. Arterial pseudoaneurysm and arteriovenous fistula develop rarely and can be minimized with meticulous puncture site planning to avoid puncture above the inguinal ligament or at the superficial femoral artery.

Some centers have adopted the routine use of arterial closure devices following chemoembolization to enable more rapid ambulation and to limit the duration of immobility needed following the procedure. Early

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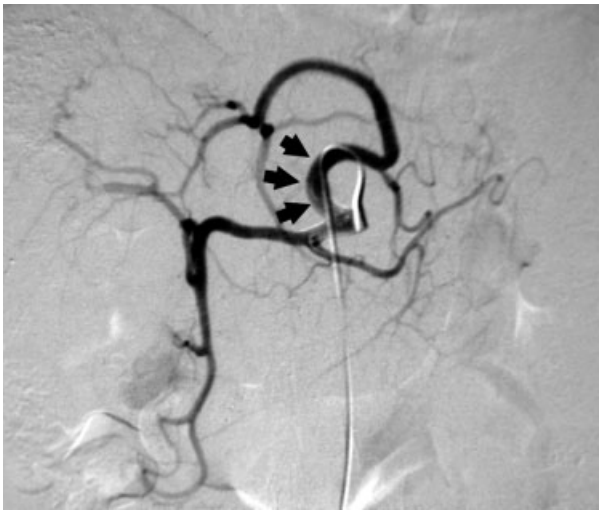

Figure 1 Iatrogenic dissection (arrows) of the left gastric artery during attempted TACE in a 48-year-old woman with hepatocellular carcinoma. A replaced left hepatic artery arises from the left gastric artery; catheterization of this vessel was not possible during this setting.

published experience with these devices in this patient population is limited but suggests that they may be used safely.[1] A comprehensive review of complications related to the use of these devices is beyond the scope of this discussion; however, the decision to use a particular device should consider the need for repeated puncture for additional TACE.

Hepatic Artery Injury

Tortuous anatomy and congenital anatomic variations in the hepatic arteries may require extensive catheter manipulation to enable access to the target artery. Advancement of a catheter against resistance or without a sufficient length of leading guide wire can produce arterial spasm, dissection, or thrombosis.[2] The routine use of microcatheters for chemoembolization may reduce the risk of arterial injury although this has not been established. Arterial spasm is usually managed with the use of vasodilators. Dissection and acute thrombosis are more problematic as they may prevent placement of a catheter into the target location and can impair inflow for delivery of the chemoembolic mixture to the tumor (Fig. 1).

Nontarget Embolization

Nontarget embolization is among the most devastating complications of hepatic chemoembolization. Nontarget embolization may occur from (1) lack of recognition of arterial supply to nonhepatic structures or (2) reflux of chemoembolic agents back along the catheter during delivery.

Nearly half of the general population harbors some form of anatomic variation of arterial blood supply to the liver, which includes replacement of the right hepatic artery from the superior mesenteric artery, replacement of the left hepatic artery from the left gastric artery, presence of a middle hepatic artery, or accessory right or left hepatic arteries.[3] These variations underscore the need for thorough visceral arteriography prior to initial chemoembolization.

Nontarget embolization of the left or right hepatic artery with chemoembolic agents may result in mucosal ulceration or perforation. Identification of these vessels is therefore of paramount importance. In patients with a large left lobe, the left gastric artery may be difficult to distinguish from a replaced left hepatic artery. Having the patient swallow effervescent granules during the procedure to produce distension of the stomach may be helpful in making this distinction, as the left gastric branches will follow the lesser curvature of the stomach. Oblique arteriography is an additional useful adjunct. The mucosa of the gastric fundus may exhibit a prominent arterial blush; when treating hypervascular tumors of the left lobe of the liver, this must be borne in mind.

The origin of the right gastric artery is variable but most commonly arises from the proper hepatic artery, the common hepatic artery, and the right hepatic artery. When visible, the embolization catheter should be positioned well beyond this vessel. Routine embolization of this vessel has been performed prior to hepatic artery infusion of chemotherapy,[4] but routine embolization of this vessel prior to chemoembolization has not been established. A balloon occlusion technique has also been described to avoid embolization of gastric branches during TACE.[5]

Nontarget embolization of the gastroduodenal artery may also result in mucosal ulceration and perforation.[6] Chemotherapy-induced ulceration may be extremely difficult to manage, even with aggressive proton pump inhibition and use of sucralfate. Nontarget embolization of pancreaticoduodenal branches of the gastroduodenal artery can result in pancreatitis. Anatomic variations of the hepatic artery with a trifurcation of the gastroduodenal right and left hepatic arteries may pose a higher risk of injury to the gastroduodenal artery. In these circumstances, meticulous fluoroscopic monitoring during chemoembolization is necessary.

Cystic artery embolization during TACE is associated with prolonged postembolization syndrome and may produce chemical cholecystitis with marked right upper quadrant pain and gallbladder wall thickening (Fig. 2).[7] When targeting right lobe tumors, positioning the catheter beyond the cystic artery with careful fluoroscopic monitoring during chemoembolization can avoid this complication in most patients. However, the distribution of tumor burden in the right hepatic lobe may require deliberate embolization of this vessel. Most patients can be treated conservatively, without the need for cholecystectomy or percutaneous cholecystostomy.[8]

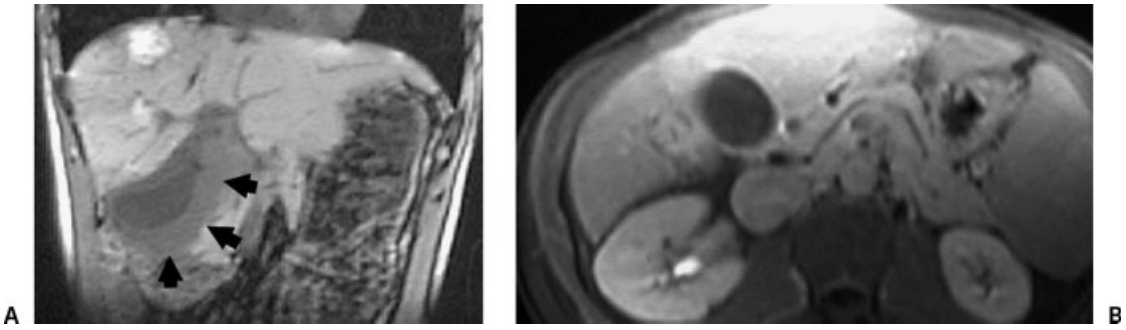

Figure 2 (A) Coronal T1-weighted magnetic resonance imaging (MRI) of a 50-year-old man with metastatic small bowel sarcoma and right upper quadrant pain 4 weeks following TACE, showing extensive gallbladder wall thickening (arrows) consistent with chemical cholecystitis. The patient was managed conservatively. (B) Transverse T1-weighted MRI obtained 3 months later showing return of the gallbladder to a normal appearance. Cholecystectomy was unnecessary.

Chemoembolization of extrahepatic collaterals may be necessary in patients with hepatocellular carcinoma (HCC) with parasitized blood supply. The inferior phrenic artery is among the most common sites of collateral supply for HCC near the dome of the liver. Embolization of this vessel predictably produces a variable degree of pleural inflammation with effusion and pleuritic chest pain (Fig. 3). Empyema formation has also been reported and mandates percutaneous or surgical drainage.[9]

Cutaneous branch embolization, such as the superior epigastric artery, can result in second- or third-degree skin burns. Arora et al described four patients with cutaneous ulceration from chemoembolization; one

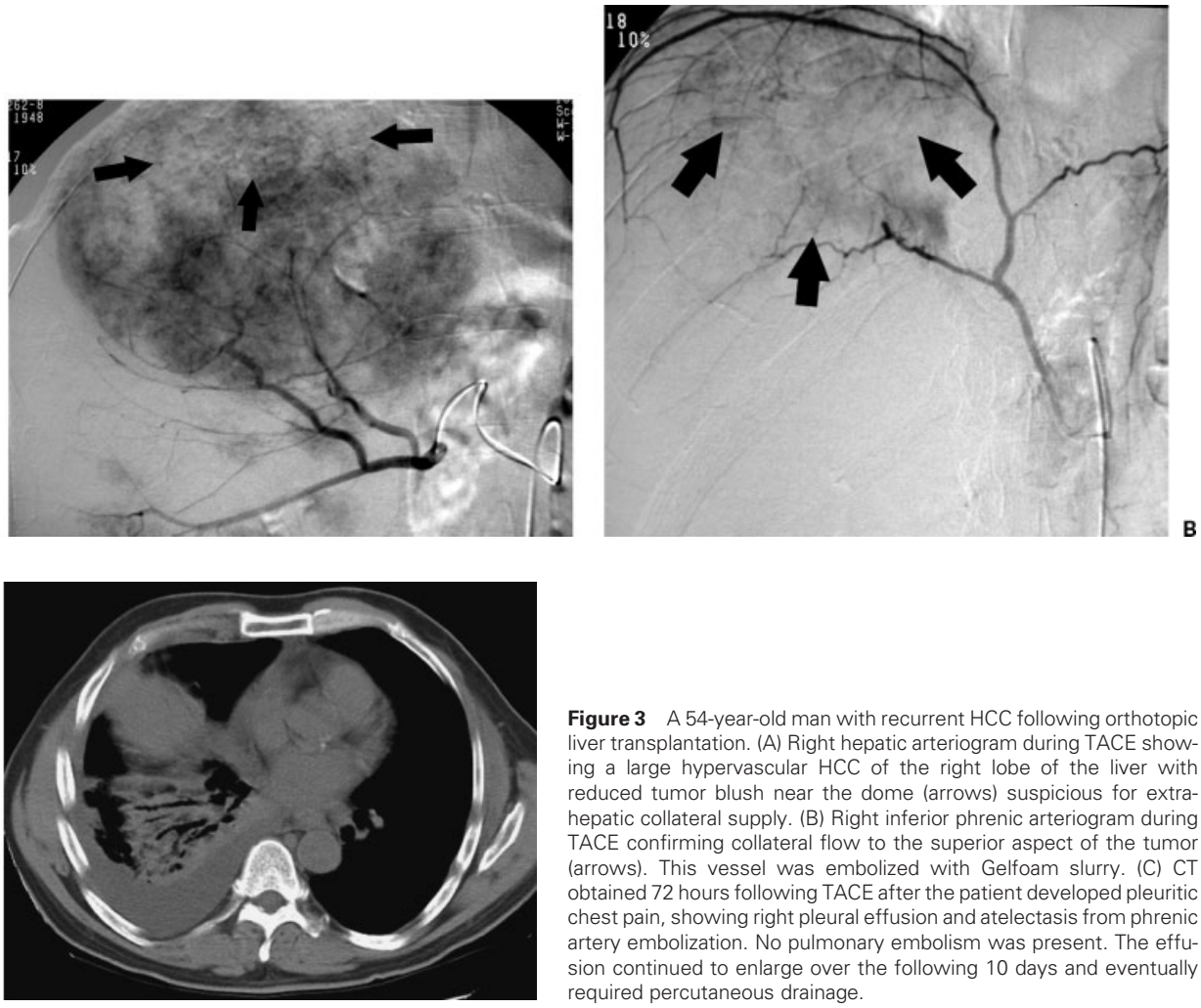

Figure 3 A 54-year-old man with recurrent HCC following orthotopic liver transplantation. (A) Right hepatic arteriogram during TACE showing a large hypervascular HCC of the right lobe of the liver with reduced tumor blush near the dome (arrows) suspicious for extrahepatic collateral supply. (B) Right inferior phrenic arteriogram during TACE confirming collateral flow to the superior aspect of the tumor (arrows). This vessel was embolized with Gelfoam slurry. (C) CT obtained 72 hours following TACE after the patient developed pleuritic chest pain, showing right pleural effusion and atelectasis from phrenic artery embolization. No pulmonary embolism was present. The effusion continued to enlarge over the following 10 days and eventually required percutaneous drainage.

patient developed a radiation burn from multiple sessions of chemoembolization, which eventually degenerated into squamous cell carcinoma.[10]

Pulmonary Embolization

Embolization of lipiodol to the lungs may occur through arteriovenous shunting or portovenous shunting.[11] This shunting may not be detectable during TACE. Mild cases of lipiodol embolization may be asymptomatic and discovered incidentally by computed tomography (CT) with high-attenuation areas of lung. Larger amounts of lipiodol to the lung can produce a chemical pneumonitis and may be fatal.[12] Lipiodol volume exceeding 20 mL has been identified as a risk factor for pulmonary embolization.[13] Patients with tumors supplied by extrahepatic collaterals appear to be at higher risk of pulmonary embolization of lipiodol.[14]

Particle embolization to the lungs may occur with smaller particle diameters. The actual threshold below which pulmonary embolization may occur has not been established, and whether microsphere embolics are at higher risk of pulmonary embolization is unknown. Brown reported three fatal cases of pulmonary embolization following the use of 40- to 120-μm trisacryl gelatin microsphere embolization shortly after these microspheres became available for use in the United States. No instances were observed using microspheres of larger diameter.[15]

NONVASCULAR COMPLICATIONS

Postembolization Syndrome

Postembolization syndrome occurs in ~90% of patients following TACE, manifested by fever, malaise, right upper quadrant pain, nausea, and vomiting. Patients and their caregivers need to be apprised of this prior to TACE and provided with adequate analgesics and antiemetics for symptom control.

Leung et al analyzed predictors of severe postembolization syndrome and found that gallbladder embolization and higher doses of chemoembolic agents correlated with prolonged postembolization syndrome.[7]

Other Toxicities

Alopecia, myelosuppression, leukopenia, and anemia may all be seen following TACE. Kessler et al reviewed common toxicity data for 149 patients following 436 TACE procedures using cisplatin, mitomycin-C, and adriamycin.[16] Anemia occurred in 2.7% of patients. In a study by Civalleri et al, myelosuppression was seen in 44% of patients following 69 sessions of TACE.[17]

Infection

HEPATIC ABSCESS AND BILOMA

The pathophysiology of biloma formation involves ischemic injury to the peribiliary capillary plexus, which is supplied by branches of the hepatic artery. As a result, the integrity of the biliary tree is disrupted with subsequent biloma formation.[18] Bacterial seeding of these bilomas can produce a hepatic abscess. Another mechanism is the development of an abscess within the necrotic center of a devascularized hepatic tumor. In either situation, patients who have chronic colonization of the biliary tree with enteric flora are at significantly higher risk of hepatic abscess formation. This includes patients with a surgical bilioenteric anastomosis, seen commonly among patients with pancreatic neuroendocrine tumors who have undergone a prior Whipple procedure. However, patients with biliary stents will have complete colonization of the biliary tree with enteral flora within 72 hours of stent placement. Prior sphincterotomy resulting in an incompetent sphincter of Oddi, can also produce biliary colonization (Fig. 4).

In the series of 157 patients from the University of Pennsylvania, hepatic abscess developed in six of seven patients with prior bilioenteric anastomosis.[19] All patients had received a standard regimen of broad-spectrum prophylactic antibiotics. Hepatic abscess developed at a mean of 19 days between TACE and hepatic abscess formation. The authors concluded this subgroup of patients mandated a more aggressive antibiotic prophylaxis regimen and closer monitoring following TACE.

In a small retrospective series, the group at Johns Hopkins observed no instances of hepatic abscess among four patients with prior bilioenteric anastomosis when an aggressive bowel preparation was used, compared with hepatic abscess in four of four patients who received

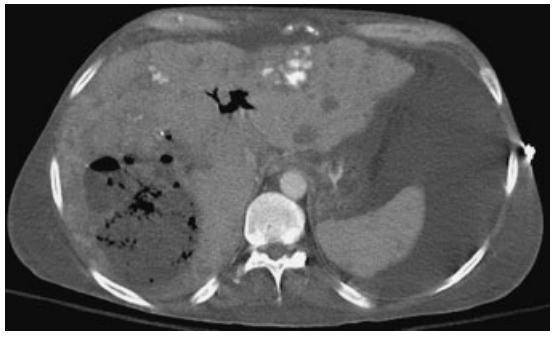

Figure 4 CT scan showing hepatic abscess in the right lobe of the liver in a 46-year-old man with metastatic neuroendocrine tumor. The patient had a history of previous biliary obstruction treated with endoscopic stent placement and had undergone TACE 5 months previously. The patient received an aggressive bowel preparation and antibiotic prophylaxis at the time of TACE. Cultures following percutaneous drainage grew coagulase-negative *Staphylococcus* and *Streptococcus constellatus*.

standard antibiotic prophylaxis.[20] At our institution, we have adopted a similar bowel preparation protocol for all patients with prior bilioenteric anastomosis who are undergoing TACE. Neomycin 1 g plus erythromycin base 1 g are given at 1 PM, 2 PM, and 11 PM the day prior to TACE. Levofloxacin and metronidazole and given intravenously while in hospital and continued orally for 2 weeks following the procedure. A review of 15 TACE procedures among six patients using this protocol found an incidence of hepatic abscess of 13%, compared with 43% among patients with bilioenteric anastomoses receiving conventional antibiotic prophylaxis.[21]

The normal post-TACE appearance of the liver on CT commonly includes gas formation within embolized hepatic tumors, with areas of fluid-dense material. These can confound the diagnosis of hepatic abscess formation, and diagnostic aspiration may be needed to establish the diagnosis of hepatic abscess.

Most patients with hepatic abscess can be successfully treated with a combination of percutaneous drainage and long-term parenteral antibiotics. In the Penn series, no patient died as a result of hepatic abscess and all responded to medical therapy and drainage. However, the natural history of untreated or undertreated hepatic abscess is associated with a high mortality.[22,23]

BACTEREMIA AND SEPSIS

Bacteremia and sepsis may be seen following TACE, even among patients without prior bilioenteric anastomosis. For this reason, most interventional radiologists use prophylactic antibiotics at the time of TACE. Reed et al observed a 2.6% rate of infection among a retrospective series of 236 patients who had received prophylactic antibiotics, compared with 11% among patients who received no antibiotics. Instances of fatal sepsis were observed in the group that received no antibiotics.[24] In our practice, we administer intravenous cefazolin and metronidazole at the time of TACE and continuing while the patient is in hospital, followed by a 5-day course of oral amoxicillin/clavulanate or ciprofloxacin.

EXTRAHEPATIC ABSCESS

Extrahepatic abscess following TACE is rare but has been reported in the spleen,[25] lung,[26] and subphrenic space.[27,28]

Biliary Strictures

Patients with biliary obstruction are at higher risk of biliary injury, with resultant stricture and biloma formation. This risk remains elevated even when patients have undergone percutaneous or endoscopic drainage of the involved segment(s) prior to TACE. Patients with extrahepatic biliary obstruction have a higher vascular permeability of the peribiliary capillary plexus,[29] which may account for this susceptibility.

Biliary strictures of the common hepatic or common bile duct will usually require percutaneous or endoscopic biliary drainage, particularly when cholangitis is present. Segmental biliary obstruction from strictures of isolated ducts may also predispose a patient to cholangitis and require drainage.

Hepatic Failure

The combination of elevated lactate dehydrogenase >425 mU/mL, aspartate transaminase >100 mL, serum bilirubin >2 mg/dL, and tumor burden $>50\%$ of liver volume is widely used as a criterion for identifying patients at high risk for hepatic failure following TACE.[30] However, these thresholds are derived from a single unpublished series and have never been systematically or prospectively studied. Consensus exists that the risk of hepatic failure is dependent on baseline hepatic synthetic function. For this reason, most interventional radiologists will not treat patients with Child C liver disease. However, liver failure may also occur in patients with Child A or B disease. In a prospective study, Huang et al observed an acute liver failure rate of 13.4% among 142 patients with HCC undergoing TACE, defined as increased Child-Pugh score ≥ 2, increase in serum bilirubin ≥ 2 mg/dL, or new ascites/encephalopathy within 2 weeks of the procedure. Among 103 patients with Child A disease, liver failure occurred in 4%, compared with 38% of 39 patients with Child B disease.[31] Among 351 patients undergoing 942 TACE procedures, Chung et al (using the same criteria of liver failure) found an incidence of acute liver failure of 15%.[32] Most of these cases were transient; permanent deterioration in liver function occurred in 5.7% of all patients.

Patients with portal vein thrombosis are at higher risk of liver failure following TACE, but these patients can still be treated when adequate collateral flow exists to the liver.[33] In our own practice, we perform TACE in a superselective fashion when portal vein thrombosis is present, using reduced amounts of the chemoembolic mixture.

Variceal Bleeding

TACE produces elevation in portal blood flow[34] and portal pressure[35] and in rare instances may precipitate variceal bleeding in patients with coexisting portal hypertension.[32,36]

Renal Failure

The risk of developing acute renal failure is multifactorial. Huo et al prospectively evaluated 140 patients undergoing TACE for acute renal failure, defined as an increase in serum creatinine >1.5 mg/dL. By this

definition, acute renal failure developed in 9% of patients; a third of these patients developed chronic renal insufficiency. Repeated TACE, severe postembolization syndrome, diabetes, and severity of underlying liver disease were identified as significant risk factors for the development of acute renal failure.[37] Kim et al reported a case of acute renal failure following TACE in a patient with normal renal function; renal failure was attributed to the combination of volume depletion from severe postembolization syndrome and contrast nephropathy.[38]

The risk of dehydration can be minimized through vigorous hydration in the periprocedural period. When administering TACE regimens involving nephrotoxic agents (e.g., cisplatin), this is particularly critical. At the University of Pennsylvania, we administer normal saline intravenously at 150 mL/h for 3 L, commencing 1 to 2 hours prior to TACE. Thereafter, half-normal saline is given at 80 mL/h until the patient resumes adequate intake of oral liquids.

Release of Neurohumoral Factors

TACE of metastatic carcinoid and other functional neuroendocrine tumors of the liver may result in massive hormonal release following the procedure. For this reason, all patients are pretreated with octreotide prior to TACE. Massive serotonin release from carcinoid tumors can produce hypertensive crisis through activation of 5-hydroxytryptamine vascular smooth muscle receptors with resultant vasoconstriction. These patients require close observation in the postprocedure period and management of blood pressure in a monitored unit may be required in the first 24 hours. The use of meperidine (Demerol®, Sanofi-Synthelabo Inc., New York, NY) should also be avoided in carcinoid patients as it inhibits serotonin metabolism and can exacerbate hypertensive effects following TACE.[39] Patients with insulinomas may require an infusion of dextrose immediately following TACE to avoid hypoglycemia, and patients with glucagonomas may require a sliding scale of insulin. Centers performing TACE for patients with neuroendocrine tumors vary in their dosage of octreotide prior to TACE; in our practice, we advocate the use of high-dose (500 μg subcutaneously) octreotide immediately prior to TACE in these patients.

SUMMARY

With careful patient selection and evaluation, major complications following hepatic chemoembolization are uncommon. However, certain at-risk patient populations exist and warrant adjustment of the TACE protocol and/or postprocedure monitoring. Awareness of these scenarios and their management are critical to performing TACE safely with optimal outcomes.

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